

FERGUSON (J.)

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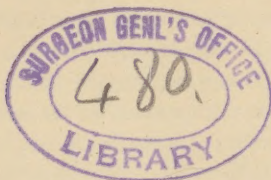
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THE subject of athetoid and choreic movements is a highly important and engrossing one. Little by little, progress is being steadily made on the morbid anatomy and pathology underlying these movements. In all conditions of brain pathology, and in the varied symptoms resulting from these morbid cerebral changes, many cases must be carefully collected and collated with each other before any definite conclusions can be deduced from the observed anatomical changes. The lesion must be limited to one special part of the brain in order that exact opinions may be formed. Should there be structural change involving two portions of the brain possessing well-known and different functions, the study becomes more complicated by the mixing of the resultant symptoms. If, however, the morbid process extends into parts of the brain of unknown, or slightly known, functions, it is quite impossible to disentangle the varying features of the symptom group, and say what is due to the pathological changes affecting one or other of the parts. Taking the contents of the cranial cavity as a whole, there

are many sections whose functions are extremely obscure. A definite lesion, then, of any part of the central nervous system, where the symptoms have been carefully noted *intra vitam*, is of the utmost importance as a means of guiding medical science one step farther in the pursuit of information bearing on the question of the localization of function, and, consequently, clearing up points in diagnosis.

It is a well-known fact that after some attacks of hemiplegia these athetoid or continuous and choreic or jerky movements come on. Another fact is that these movements may continue after the paralysis has passed away. From this we conclude that the source of irritation must be located somewhere else than in the motor areas or tracts of the brain, but yet so placed as to indirectly affect these. W. R. Gowers, in his work, says: "Regarding the nature and position of the disease causing these disorders of movement, we have as yet but little pathological evidence. The symptom is observed after recovery from the paralysis, and hence in patients who for the most part live on and pass out of observation. But two ætiological facts are of great significance. The first is, that these disorders of movement are far more frequent after cerebral softening from vascular occlusion than after cerebral hæmorrhage. The second is that they follow hemiplegia far more frequently when this comes on in infancy or childhood than when it comes on in adult life. Regarding the seat of the lesion which gives rise to these symptoms, the facts are too few to permit of accurate generalization. In most of the recorded cases the disease has been situated either in or outside the optic thalamus. Since the optic thalamus is not in the motor path, disease limited to this must produce the symptoms indirectly by disturbing the function of the motor cortex."

At a meeting of the American Neurological Society Dr. G. M. Hammond reported the pathological findings in the

original case of athetosis on which Dr. W. A. Hammond's description of athetosis was based. The portion involved in the lesion was a lengthy one in the antero-posterior direction, parallel in its short axis with the internal capsule. Its posterior end had invaded the stratum zonale of the thalamus in its posterior half of the internal capsule. In its anterior extension it had crossed the capsule, invading the posterior third of the outer articulus. The author called attention to the fact that the motor tract was not implicated in the lesion, and argued that this case was further evidence of his theory that athetosis was caused by irritation of the thalamus, the striatum of the cortex, and not by a lesion of the motor tract. Dr. Spitzka reported a case in which the lesion was found in the same situation as in Dr. Hammond's case. Dr. E. C. Seguin also gave a paper on athetoid and choreic movements in a patient. The post-mortem revealed a glioma of the left thalamus and internal capsule, the movements having been on the right side. The author's views were that all cases of athetoid and choreiform movements following hemiplegia were due to injuries of the thalamus and adjacent capsule.

To the foregoing cases I shall now add the details of one that was under my own observation in which the athetosis was well marked and was not clouded by any other motor or sensory disturbances.

The patient was a tall, thin man, of about thirty years. On December 3, 1890, I saw him for the first time, and made the diagnosis of diabetes mellitus. For this he was under treatment to the time of his death, in January, 1892.

In the early part of July, 1891, he was taken ill with the continuous movements of his right arm and leg, which lasted unabated to the evening of his death. The movements were slow and continuous, rather than short and jerky; and in these respects more closely resembled athetosis than chorea. The

movements of the arm, hand, and fingers were extensive. The arm would be carried with a steady swinging motion round behind the back, up over the neck and head, and round to the front again. Flexion, extension, abduction, and adduction of the fingers would succeed each other in endless rotation.

There were no sensory disturbances other than the existence of neuralgic pains. The knee-jerk was wholly gone on both sides, and sexual power was equally lost.

The post-mortem was made about twelve hours after the death of the patient. The brain, cerebellum, medulla, and pons were removed for examination. The left thalamus opticus presented the appearance in its substance of an old but distinct blood clot. Around this clot there were evidences of degenerative changes, the thalamus being much reduced in consistency. The changes in the left thalamus were both gross and minute. The above conditions were visible to the naked eye. Under the microscope both fresh and hardened sections revealed abundant signs of degenerative changes. This was very patent in the region of the pulvinar. No definite histological characters could be made out in either the central gray matter or in the stratum zonale, due, no doubt, to the softening caused by the irritation of the clot.

The commissura mollis was not invaded, and the right thalamus perfectly normal. The disease had not extended outward so as to involve the internal capsule, with the slight exception of a very small amount of degeneration that extended for some distance upward in the most anterior part of the capsule; or in that part which constitutes the anterior peduncle of the thalamus, by means of which it is brought into direct connection with the frontal lobe. Along the inferior peduncle of the thalamus which joins it with the temporal lobe, degeneration could be traced for only an exceedingly short distance. No pathological changes could be discovered in the lenticular or caudate nucleus. The floor of the fourth ventricle was examined, but with negative results.

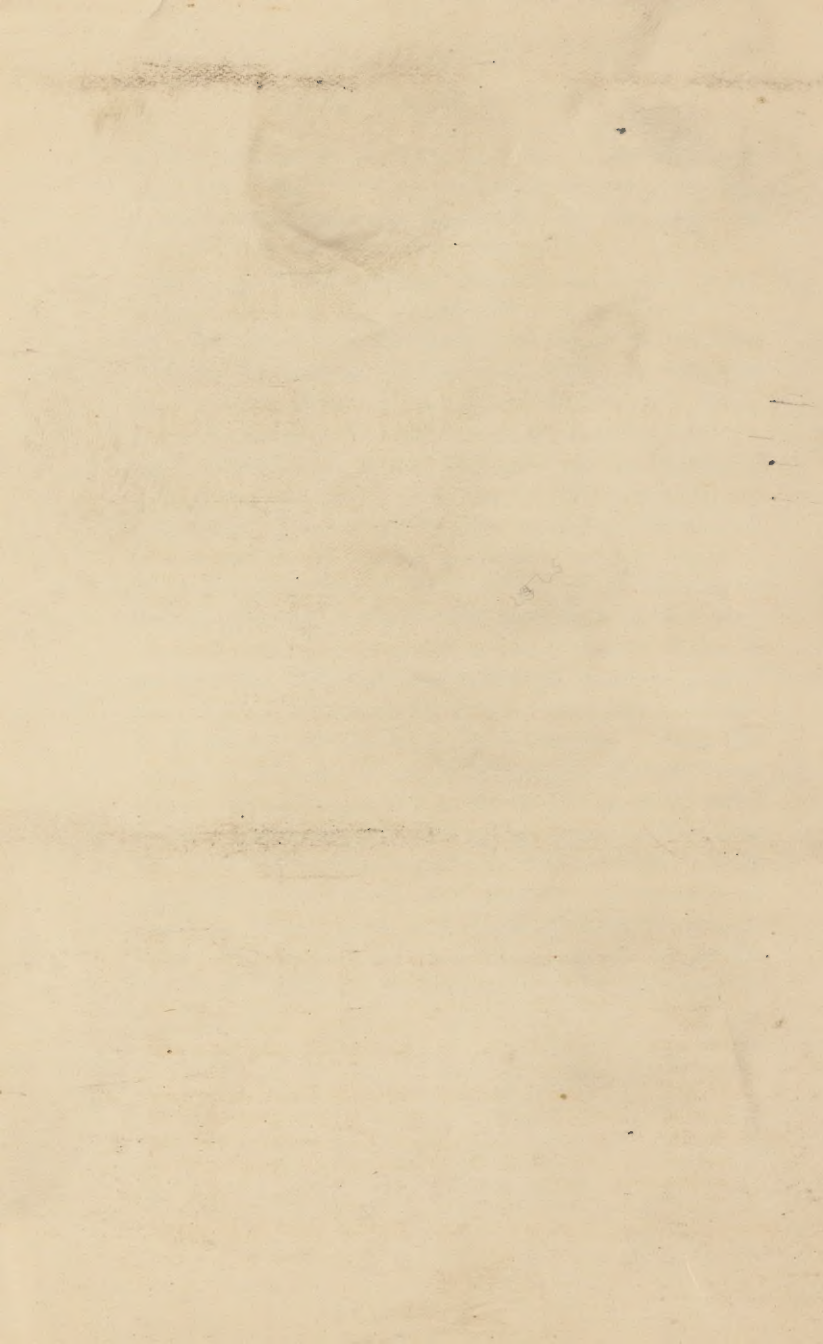
In this case there are several features of extreme importance. First, we have the positive existence of athetosis.

In the second place, the occurrence of both gross and microscopic changes in the left thalamus. Thirdly, the entire absence of any diseased conditions in the motor tract, either cortical, capsular, crustal, or pyramidal. And, finally, the important fact that there were no sensory derangements.

How are we to account for the athetoid and choreoid movements in these cases of lesion in the thalamus. It would be somewhat beyond the object of this paper to quote from all the cases that have been reported to show the constancy of disease in the thalamus in such cases. Gowers believes that disease limited to these ganglia causes the movements by indirectly irritating the motor cortex. Ross says, in his work on nervous diseases, that it is not easy to give reasons why choreiform movements are so liable to occur when the lesion is situated in the region of the posterior external optic artery. Two probable explanations of these clonic spasms suggest themselves. The first is that fibers connecting the cerebrum with the cerebellum are injured by these lesions, so that the regulation between the tonic (cerebellar) and clonic (cerebral) actions of the body is lost. The second is that the spasms are caused by partial injury of the fibers of the pyramidal tract, which regulate the fundamental movements of the body. The fundamental actions are regulated from the convolutions near the longitudinal fissure. Fibers from these convolutions would descend on the inner side of the internal capsule, and consequently on the side in contact with the thalamus. They would therefore be more liable to injury, or irritation, in disease of the thalamus than the fibers lying more external.

With regard to these views of Dr. Ross, it may be remarked that though the latter might explain some of the cases, it could not explain the one I have recorded in this paper, as the internal capsule was entirely free from disease of any kind, and therefore the fibers governing the funda-

mental movements could not have been in any way interfered with. The former view is negatived by my case also, as there was no disease or degeneration in the peduncular fibers leading to or from the cerebellum. It is not possible to say whether or not Gowers's view is true, as the thalamus is so extensively connected with the cerebral cortex. My own view is that the gray matter of the thalamus must be regarded as cortical in function, and therefore an originator of nervous energy, and not merely a transmitter by means of the numerous bundles of fibers connecting it with other portions of the central nervous system. If it originates movements, when diseased, they partake of the purposeless character of athetosis or chorea. In the case now recorded there was no motor paralysis; but the athetoid movements were greatly increased by all attempts at definite voluntary actions. This fact alone, taken in conjunction with the other considerations of positive disease in the thalamus, while there was no disease elsewhere, goes to show that the thalamus must have some regulating function to fill toward our voluntary motor impulses. When this regulating function is lost, we are able to originate movements, but no longer able to so harmonize these movements as to execute a regulated and purposive work by their aid. I would therefore regard the thalamus opticus as an originator of movements; and, secondly, as a regulator, in some way, of the movements emanating from other motor areas. The thalamus may have other functions besides the above; but I think the two mentioned can be safely inferred from my case.





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